Case Reports

Pericarditis With Effusion and Tamponade Complicating Left Subdiaphragmatic Abscesses

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ALTHOUGH NONSUPPURATIVE PLEURAL EFFUSIONS have been associated with subdiaphragmatic abscesses, analogous pericardial effusions as sequelae of subdiaphragmatic abscesses are not described in standard references.[1-4] Manual searches of *Quarterly Cumulative Index* from 1916 through 1926, of *Quarterly Cumulative Index Medicus* from 1927 through 1956, of *Current List of Medical Literature* from 1957 through 1959, and of *Cumulated Index Medicus* from 1960 through 1965 and a computerized search of *Cumulated Index Medicus* from 1966 through 1986 uncovered no references of direct relevance. We report four cases of sympathetic pericardial effusion associated with left subdiaphragmatic abscesses. In two cases, cardiac tamponade with cardiopulmonary arrest occurred, with a fatal outcome in one.

Patients and Methods

We reviewed the charts of all patients discharged from three Charlotte, North Carolina, hospitals—Charlotte Memorial Hospital, Presbyterian Hospital, and Mercy Hospital—from July 1984 through June 1986 with a diagnosis of peritonitis or intra-abdominal abscess (ICD-9-CM code 567.2).[5] Cases were evaluated for the presence of pericarditis, pericardial effusion, or tamponade. Evidence of pericardial disease by auscultation, echocardiogram, pericardiocentesis, surgical procedure, or autopsy was required for inclusion.

Statistics

The frequency of pericardial disease with right and left subdiaphragmatic abscesses was compared using the Fisher's exact test.*

Results

Of 182 cases of intra-abdominal infections, 4 of 20 cases of subdiaphragmatic abscess had associated pericardial disease (Figure 1). Of eight cases of left subdiaphragmatic abscess, five had an associated left pleural effusion, four of which also had recognized pericardial involvement. Of 12 cases of right subdiaphragmatic abscess, 3 had right pleural effusions or empyema, 1 had right and left pleural effusions, and none had recognized pericardial involvement. Eight cases of intra-abdominal infection not localized to the subdiaphragmatic spaces had pleural effusions, but no cases of pericardial disease complicating intra-abdominal infections other than subdiaphragmatic abscesses were identified.

Reports of Cases

CASE 1. The patient, an 82-year-old-woman, presented with left chest pains. She had had many abdominal operations and had congestive cardiomyopathy, sick sinus syndrome treated with a permanent pacemaker, and recurrent urinary tract infections. Three months previously, left upper quadrant pains and low-grade fever had led to an admission to hospital; an *Enterobacter* urinary tract infection was treated with the administration of doxycycline. One month before this admission, nitrofurantoin was given after urethral dilatation.

Three days of epigastric and left lower chest pains, nausea, vomiting, and diarrhea prompted a readmission to hospital. There was pronounced epigastric and mild left costovertebral angle tenderness. A chest x-ray film showed cardiomegaly. A barium enema film showed diverticula. Abdominal ultrasonography and intravenous pyelography were normal. Giardiasis diagnosed by endoscopic duodenal aspiration was treated with the administration of metronidazole hydrochloride. Fevers to 39.5°C (103°F) began.

Pericardial and left pleural rubs were noted on hospital day 3. Thoracentesis of left pleural fluid showed an exudate with a pH of 7.1, an amylase level of 40 units per dl (400 units per liter; normal for serum, 15 to 200 [150 to 2,000]), a protein level of 4.1 grams per dl (41 grams per liter), and a leukocyte count of 1,000 per μl (1.0×10^9 per liter). Echocardiography showed a small pericardial effusion. A computed tomographic (CT) scan of the abdomen suggested a splenic infarction. *Escherichia coli* was cultured from a percutaneous splenic aspirate. Therapy with cefoxitin was begun; a splenectomy confirmed the diagnosis of splenic infarction with abscess, which was densely adherent to the left subdiaphragmatic surface. Her recovery was uncomplicated.

CASE 2. The patient, a 16-year-old woman, had had abdominal cramps for three days. She had right lower quadrant guarding and a temperature of 38.3°C (101°F). A regimen of cefoxitin was begun. A ruptured appendix with a local abscess was found at laparotomy. Drains were placed, and gentamicin sulfate was added to her regimen. She had persistent fevers, so cefotaxime sodium and clindamycin were given. On postoperative day 6, a pericardial rub and a left pleural effusion developed; a moderate pericardial effusion was shown echocardiographically. Later, mezlocillin and metronidazole were given. Turbid pleural fluid removed by thoracentesis clotted, and cultures were sterile. On days 8 and 14, CT scans showed a left subdiaphragmatic abscess and increasing pericardial and bilateral pleural effusions. Cultures of abscess fluid obtained by percutaneous drainage were sterile.

On day 15, she became dyspneic, tachypneic, and agitated with hypotension and a paradoxic pulse of 20 mm of mer-

*The statistical analysis was done by Rachel Ballard-Barbash, MD, of the Nalle Clinic.

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cury. She had periorbital edema, distended jugular veins, and distant heart sounds. An electrocardiogram showed low voltage. The central venous pressure was too high to measure with a manometer. A cardiac arrest from tamponade was treated successfully, and pericardiocentesis of 250 ml of serosanguineous fluid improved her hemodynamics. Fluid analysis showed a protein level of 3.9 grams per dl (39 grams per liter), a glucose level of 100 mg per dl (5.4 mmol per liter), and an erythrocyte count of 1.74×10^6 per μl (1.74×10^{12} per liter). The fluid leukocyte count was 2,600 per μl (2.6×10^9 per liter) with a differential count of 36 segmented neutrophils, 39 bands, 4 lymphocytes, 5 eosinophils, 4 metamyelocytes, 1 myelocyte, 1 tissue cell, and 8 nucleated erythrocytes. Cultures for bacteria, mycobacteria, and fungi were sterile. After the arrest, she had a transient coagulopathy. An antinuclear antibody titer of 1:1,280 in a speckled pattern was attributed to the severe infection. On day 24, a pericardiectomy revealed 300 ml of bloody pericardial fluid and fibrinous pericarditis, and a laparotomy showed a resolving left subdiaphragmatic abscess. *Candida* sepsis was treated with the administration of amphotericin B. Further recovery was uneventful. During 18 months of follow-up, no evidence of systemic lupus erythematosus has developed.

CASE 3. A 27-year-old woman had left lower quadrant pain for one day. She had a distant history of splenectomy after a motor vehicle accident, and she had been treated for pneumonia one month previously. On hospital admission, she had a blood pressure of 100/60 mm of mercury, a pulse of 140 beats per minute, and a temperature of 40°C (104°F). The abdomen was rigid and had diffuse rebound tenderness. Therapy with cefazolin, clindamycin, and gentamicin was begun; later, cefoxitin was given. At laparotomy a pelvic abscess was noted, and blood and peritoneal cultures grew *Streptococcus pneumoniae.*

On hospital day 3, a chest x-ray film showed an enlarged cardiac silhouette and left pleural effusion. An electrocardiogram was normal on day 4. Persistent fevers by day 7 led to ultrasonographic and CT scanning, showing a left subphrenic abscess and minimal pericardial fluid. A percutaneous aspirate of 75 ml of pus from the abscess showed many neutrophils but no organisms on Gram's stain; cultures were sterile. An echocardiogram showed a pericardial effusion. She recovered smoothly. On day 20, a repeat echocardiogram showed a small pericardial effusion. After late resolution of her fever, she recovered smoothly.

CASE 4. The patient, a 34-year-old man with hypertension and recurrent familial pancreatitis, had abdominal pain for four hours. He was afebrile and had a blood pressure of 160/120 mm of mercury. Generalized abdominal distension without rebound tenderness was present, and bowel sounds were absent. A serum amylase level was 104 units per liter (normal 25 to 115).

On hospital day 1, a right internal jugular venous catheter was inserted, and a serum amylase level was 418 units per liter. On day 2, the administration of parenteral nutrition was begun. On day 4, a left subclavian venous catheter was placed and a fever to 39°C (102.2°F) developed. Cefazolin therapy was begun but was changed to imipenem-cilastatin sodium. On day 5, the left subclavian catheter was removed because of insertion site cellulitis and a right internal jugular venous catheter was inserted. Blood cultures grew *Staphylococcus aureus.* The imipenem-cilastatin therapy was stopped and cefazolin therapy resumed.

On day 9, he became dyspneic and hypotensive. An electrocardiogram showed a sinus tachycardia. Pulmonary embolism was suspected, and heparin was given briefly pending

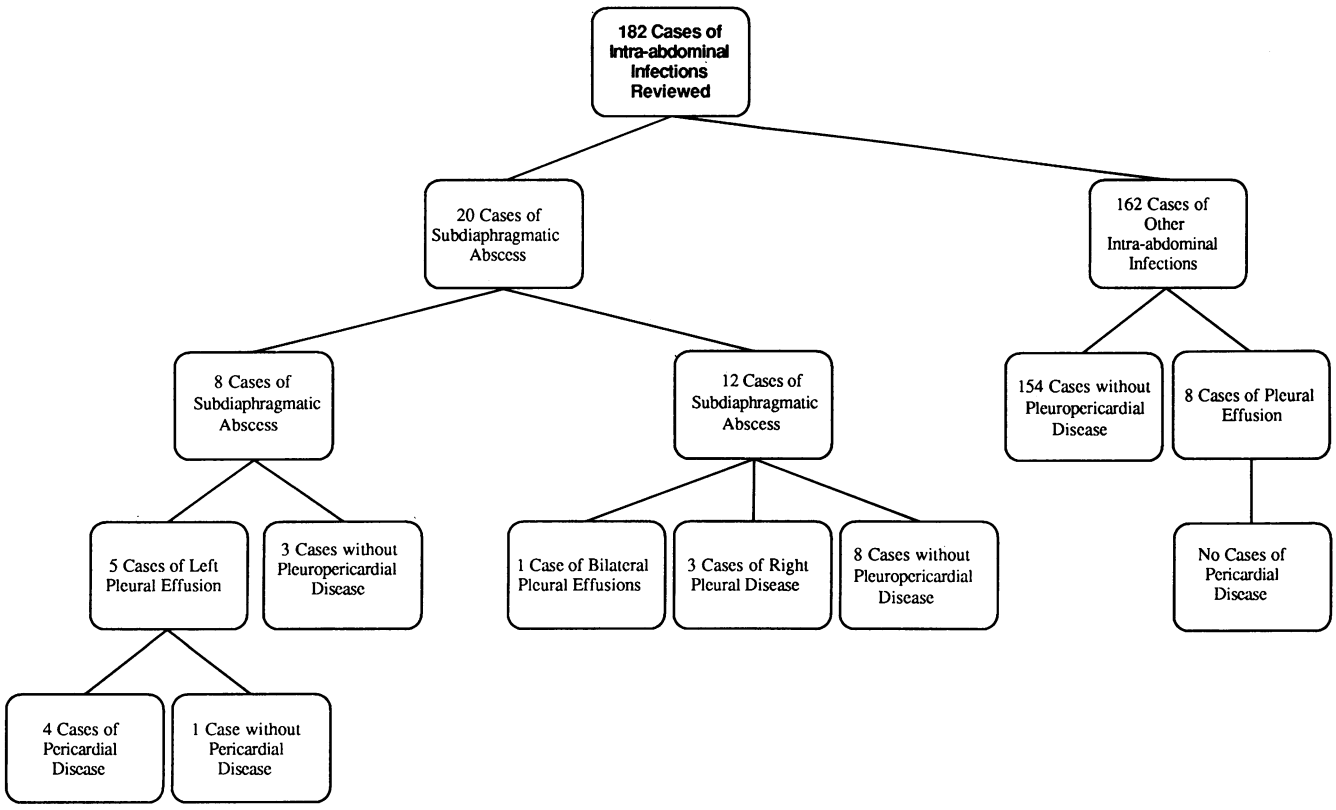

Figure 1.—The diagram shows 182 cases of intra-abdominal infections reviewed.

the outcome of a radionuclide ventilation-perfusion scan, which was normal. Cardiac tamponade was diagnosed echocardiographically. A cardiac arrest occurred as an emergency pericardial window was created, releasing tense fluid. The clear amber pericardial fluid had a protein level of 0.7 grams per dl (7 grams per liter), a specific gravity of 1.016, a glucose level of 2,046 mg per dl (109 mmol per liter) with a blood glucose level of > 400 mg per dl (> 21 mmol per liter), and an amylase level of 0 units per liter. No fluid cultures or cell counts were done. No cardiac perforation was found. The antibiotic regimen was changed to nafcillin sodium and later to vancomycin hydrochloride with tobramycin. The patient died on day 20 of ischemic encephalopathy. An autopsy revealed pancreatic fibrosis and atrophy, pulmonary emboli, fibrinous pericarditis without cardiac perforation, a left pleural effusion, and a 1-cm perforated gastric ulcer with a loculated left subdiaphragmatic abscess.

Discussion

The classic review of subdiaphragmatic abscesses by Ochsner and DeBakey from 1938 acknowledged thoracic complications of subdiaphragmatic abscesses, including empyema, bronchial fistula, and pericarditis.[6] In that preantibiotic era, however, most, if not all, of these complications probably were suppurative, occurring from direct penetration or perforation of infection through the diaphragm or from bacteremic colonization of effusions.[7] To the best of our knowledge, in the past 50 years, with one possible exception,[8] the pericarditides reported with subdiaphragmatic abscesses have been suppurative in nature and have occurred with infections penetrating through the diaphragm. Our four cases support an association between nonsuppurative, sympathetic pericardial effusions and left subdiaphragmatic abscesses. In contrast to none of 12 cases of right subdiaphragmatic abscess we reviewed, 4 of 8 cases of left subdiaphragmatic abscesses had pericardial findings. The association of pericardial disease with left subdiaphragmatic abscesses was statistically significant ($P = .014$), although we cannot be certain that patients with right and left subdiaphragmatic abscesses were equally likely to be evaluated for pericardial disease.

In our series, pericarditis and pericardial effusions were recognized four to nine days after the onset of symptoms of abdominal infection. Symptoms included chest pain and dyspnea. Cardiac tamponade occurred in two cases and was ultimately fatal in one. In only one patient (case 3) was the pericardial process apparently silent and discovered fortuitously by sensitive technologic means, such as CT scanning. Even with early detection of pericardial fluid in case 2, however, the potential for cardiac tamponade was underappreciated and that catastrophe was not averted. Two patients (cases 1 and 3) had pericardial fluid shown by echocardiography. The two other patients (cases 2 and 4) had pericarditis with effusion confirmed by pericardiocentesis, pericardiectomy, or autopsy.

In none of the four cases was there evidence of purulent pericarditis. Pericardial fluid cultures were negative in case 2, although antibiotics had been given. The patient had an elevated antinuclear antibody titer, but 18 months later she had no evidence of autoimmune disease that might have caused the pericarditis. The patient in case 4 had an elevated glucose level in the pericardial fluid, possibly suggesting central venous catheter perforation, but no evidence of cardiac perforation was found at operation or at autopsy. Although patients 2 and 4 had bleeding diatheses during their illnesses, pericardial fluid found in each case was not consistent with hemopericardium. None of the four patients showed pancreatic pleuropericardial effusions.

The pathophysiologic mechanism for sympathetic pericarditis with effusion in the setting of subdiaphragmatic abscess is unknown. Pleural effusions are recognized to occur from subdiaphragmatic infections on either the right or the left.[6] These pleural exudates may be caused by changes in capillary permeability or lymph flow in the diaphragm induced by local effects of inflammatory products.[2,6,9,10] We hypothesize a similar mechanism for the pericardial findings in our series. The proximity of subdiaphragmatic inflammatory processes to the pericardium overlying the left diaphragm may be a key factor in determining whether pericardial complications develop. With relatively little liver mass adherent to the left subdiaphragmatic surface, abscesses there have exposure to a large area over which the pericardium lies.[11]

Each patient presented with infection, not with pericardial disease. With treatment with potent, broad-spectrum antibiotics and with surgical interventions, mortality from uncontrolled infection was nil. The indirect pericardial complications of the left subdiaphragmatic abscesses in our series were serious, however, posing life-threatening risks and requiring costly and invasive procedures for management.

Conclusion

In patients with subdiaphragmatic abscesses, early recognition of sympathetic pericarditis with effusion may be lifesaving. Especially if a left pleural effusion is present, evidence of pericarditis should be aggressively sought by bedside examination, echocardiography, abdominal ultrasonography, or CT scanning. Conversely, subdiaphragmatic abscesses should be sought when pleuropericardial exudates are otherwise unexplained.

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